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Case Report

Case Series of Collagenous Gastritis in Pediatrics with Review of the Recent Literature

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Abstract

Collagenous gastritis is a rarely diagnosed disorder in pediatrics. The pediatric onset type typically presents with abdominal pain and anemia and the adult onset type with diarrhea. We present 2 new cases of collagenous gastritis. The first case presented with abdominal pain but no anemia. The second case presented with diarrhea as seen in the adult onset form and found to have celiac disease. This is the second report in a pediatric patient with collagenous gastritis and proven celiac disease.

ABBREVIATIONS

CG: Collagenous Gastritis

INTRODUCTION

Collagenous gastritis is a rare disorder in all age groups. There have been less than 25 cases reported in children [1]. The histologic appearance of CG is similar to that of collagenous colitis. In CG the antrum or body of the stomach or both show irregular thickened sub epithelial collagen band, often discontinuous, ranging from 15 um to 120 um. There are entrapped dilated capillaries and inflammatory cells within the collagen band. In addition, there is a chronic lymphocytic infiltrate of the lamina propria; eosinophilic infiltrate can be prominent and focal mild acute inflammation can be seen. The overlying surface epithelium shows injury with flattening and focal detachment of the epithelium and may or may not be associated with intraepithelial inflammatory cells, particularly lymphocytes [2,3]. The endoscopic appearance of CG ranges from nodular to inflamed gastric mucosa.

There is reported to be a childhood form of CG and a somewhat different adult version [4]. Abdominal pain with or without iron deficiency anemia is the typical childhood presentation. The adult form is associated with other autoimmune processes and celiac disease [5]. The adult patients may also have collagenous colitis. In contrast to adult onset CG the pediatric version typically involves only the stomach.

We present two pediatric cases of collagenous gastritis. The first case was a 3 years old boy with type 1 diabetes mellitus and the typical childhood complaint of abdominal pain seen with CG. The second case a 15 years old female with abdominal pain and anemia who was also diagnosed with celiac disease, the pattern typically seen in the adult version of CG but reported only once in pediatrics [6]. A review of the prior cases of CG reported in childhood will be undertaken and compared to our cases.

CASE PRESENTATION

Case Report 1

A 3 years old male presented with a one month history of vomiting and upper abdominal pain. He was diagnosed with Type 1 diabetes mellitus one year prior to his gastrointestinal complaints. The only medication use noted was insulin. Growth and development were normal. Family history was positive for a maternal uncle who has Type 1 diabetes mellitus and Crohn's disease. His physical examination was normal, without hepatosplenomegaly. He had normal abdominal ultrasound, MRI of the head, gastric emptying scan and celiac serology, including Deaminated Gliadin Peptide IgG antibody (DGP IgG), Deaminated Gliadin Peptide IgA antibody (DGP IgA), Anti-Human Tissue Transglutaminase IgA ELISA (TTG IgA), and Anti-Endomysial IgA IFA (EMA IgA). His serum IgA level was within normal range for age. His CBC with differential and liver function panel was normal. Prior to any medication trials he underwent esophagogastroduodenoscopy. Endoscopy revealed gastric nodularity of the antrum extending into the body of the stomach. Biopsies from that region noted focal thickening of the sub epithelial collagen layer (Figure 1), confirmed by trichrome stain (Figure 2). The lamina propria contained an inflammatory cell infiltrate, consisting of lymphocytes, plasma cells and eosinophils. Colonoscopy was not done. The patient was started on lansoprazole 15mg daily. His emesis and pain resolved in a few weeks. Nine months after the initial endoscopy a repeat esophagogastroduodenoscopy revealed normal appearing gastric mucosa. Biopsies from the previous abnormal areas were now normal.

Case Report 2

A 15 years old female presented with a one year history of abdominal pain and diarrhea. It was also noted that she had minimal linear growth over that year. She was treated intermittently with minocycline for acne for 18 months prior to her presentation, and did not take any other medications. There was no family history of celiac disease or inflammatory bowel disease. Her physical examination was normal without hepatosplenomegaly. Her hemoglobin was 9.6 g/dl and hematocrit 29.7 %. Total iron was 30 ug/dl, iron binding capacity 399 ug/dl and iron saturation low at 8%. EMA IgA screen was positive (titer 1:20, with normal <1:5), TTG IgA was 10 U/ml (normal <5), and total serum IgA was normal, 117 mg/dl. (normal range for age 44-441). On esophagogastroduodenoscopy she was found to have several small ulcers in the body of the stomach. Gastric biopsies noted focal thickening of the sub epithelial collagen layer, confirmed by trichrome stain. The duodenal biopsies were normal. Colonoscopy was not done. She was started on omeprazole 20mg daily and iron. Within one month she had resolution of her abdominal pain and diarrhea. Ten months later the EMA IgA remained positive, and TTG IgA was also still positive at 14 U/ml. Her hemoglobin was normal 13.1gm/dl. Her iron panel was normal. She underwent her second esophagogastroduodenoscopy 10 months after the initial one and was found to have diffuse gastritis. Duodenal biopsies showed patchy villous blunting, lamina propria plasmactyosis and intraepithelial lymphocytosis. Trichrome stain of the gastric tissue showed improvement from her first biopsies, with remaining minimal focal thickening of the collagen table.

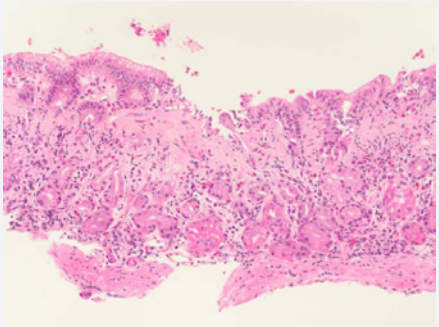

Figure 1 H and E stain (magnification 20 x): A thickened sub epithelial collagen band is seen beneath the surface epithelium, which is focally detached. The lamina propria shows chronic inflammation including eosinophils.

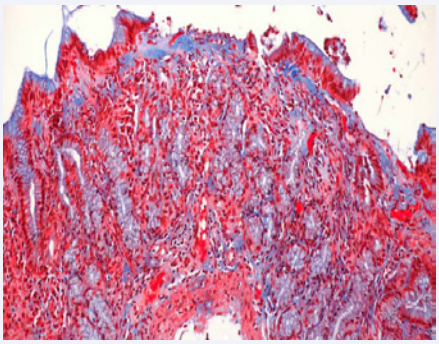

Figure 2 Masson trichrome stain (magnification 20x) highlighting the thickened collagen band.

DISCUSSION

Collagenous gastritis is rarely diagnosed in children [1,6]. Its etiology is unknown. Celiac disease has been reported once before with CG in pediatrics [7]. Helicobacter pylori (H. pylori) gastritis has been associated with collagenous gastritis though not seen in either of our cases (H.pylori immunostains were negative) [8]. Our first patient had both symptomatic resolution of his presenting symptoms of abdominal pain and emesis as well as histologic resolution. Whether this was due to the use of a proton pump inhibitor or just the natural history of the illness is not clear. The second patient was not initially treated with a gluten free diet. She received enteral iron which resolved her anemia. She was also placed on a proton pump inhibitor. Her abdominal pain resolved on this treatment without institution of a gluten free diet. Follow up biopsies in this patient did reveal

persistence of her gastritis and now clear evidence for celiac disease. She was placed on a gluten-free diet but lost to follow up.

Collagenous gastritis was first described by Drs. Coletti and Trainer in 1989 [9]. The patient reported by Dr. Coletti was 15 years old who had abdominal pain and iron deficiency anemia, the typical childhood presentation. Since the first reported cases of CG in pediatric patients there have been 23 other cases reported in children with the most recent publications in 2009-2013 [1,6,10]. We report one male and one female patient. Seventeen of the 24 previous reported patients were female [1]. Most patients present with anemia but only one of our cases had anemia [11]. Proton pump inhibitors are the most frequent treatment modality. A variety of treatments have been used to treat CG. These include proton pump inhibitors, H2antagonists, corticosteroids, sucralfate, 5-aminosalicylate (5-ASA) drugs and hypoallergenic diets. There has been no consistent reported effectiveness of any of these treatments. Both of our patients had symptom resolution with the use of a proton pump inhibitor though one could argue that the symptoms abated on their own since there have been reports of spontaneous resolution of symptoms. Pediatric CG appears to follow a benign course. Though the histologic lesion may persist, patients have been reported becoming asymptomatic as was noted in both or our patients [8,12,13]. In adult onset CG several systemic autoimmune disorders have been noted [14]. Their prognosis would predictably be more guarded. One of our patients has Type 1 diabetes mellitus the other celiac disease. There was no clinical evidence for other autoimmune diseases in either of the patients in this report.

For treatment purposes we recommend iron supplementation when indicated by the appropriate testing. Coexisting H. pylori infection should be treated, especially since H. pylori colonization in gastric mucosa may impair iron uptake and increase iron loss, potentially leading to iron deficiency anemia [15-17]. Patients should be followed for development of autoimmune disorders. Strong consideration should be given toward colonoscopy due to the concern regarding coexisting collagenous colitis. Treatment of abdominal pain with acid suppression to provide symptomatic relief seems a reasonable first treatment step. Due to the scarcity of children with this disorder treatment beyond acid suppression such as use of steroids should be reserved for patients with intractable symptoms such as abdominal pain and anemia.

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